solutions for the long dwell. In contrast, we compared icodextrin with glucose 1.36% in patients without clinical signs of overhydration, of whom the great majority had significant residual renal function (mean rGFR at start 4.8 ± 3.2 mL/min). At that time, 1.36% solutions were used in our clinics for the long dwell in patients with significant residual diuresis without clinical signs of overhydration.

In search for an explanation for the discrepancies between the two studies, we hypothesized that icodextrin might have led to underhydration in some of our patients. Therefore, we compared the decline in rGFR between patients who were underhydrated at the end of the study and those who were not. Underhydration was defined as a normalized ECW (ECW:height) below the 10th percentile of the stable renal transplant patients studied in [1] [<7.8 L/m in males and <7.0 L/m in females].

Four of the 19 patients in the icodextrin-treated group who completed the study fulfilled this criterion. Compared to the 13 patients treated with icodextrin who were not underhydrated after completion of the study, the fall in rGFR tended to be larger [−3.2 ± 2.4 mL/min vs −1.0 ± 1.6 mL/min; \( P = 0.055 \)]. When the underhydrated patients were excluded from analysis, the decline in rGFR between patients treated with icodextrin and the control group was comparable [−1.0 ± 1.6 vs −0.6 ± 0.8 mL/min; \( P = 0.6 \)].

In conclusion, the decline in rGFR observed in our previous study after treatment with icodextrin may have been due to underhydration in a minority of patients. Given the limited number of patients in whom underhydration was diagnosed, this assumption needs to be confirmed. However, when using icodextrin in patients with significant residual renal function without clinical signs of overhydration, objective assessment of fluid status may be helpful in defining treatment targets [4].

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REFERENCES


Arterial stiffness in patients with kidney transplantation

To the Editor: We read the recent article by Bahous et al demonstrating tobacco consumption and acute rejection modulates both aortic stiffness and renal functional deterioration after kidney transplantation [1]. We wish to raise several points that can be considered.

Primary end points, including doubling serum creatinine (4 patients) and/or new cardiovascular events (9 patients), which are both independent situations resulting from different factors, seem to be confusing. In this study design, one cannot properly expect that the pulse wave velocity (PWV), mean 54.1 months after the transplantation, can be used as an indicator of the cardiovascular disease (CVD) after the transplantation. We do not know the level of PWV before the transplantation; the patients dying after the transplantation did not have the results (12 patients), which could affect the analysis. Also, 9 patients had new CVD, and 6 of them had previous CVD. It could have been important to see these patients’ PWV results (9 patients). It is not logical to take serum creatinine into analysis of primary end points (according to the definition of primary end points). However, it could be interesting to see whether kidney function, as a risk factor for CVD, might be also a risk factor for CVD after the transplantation [2].

Tobacco consumption was given in Table 1. However, how many patients were using tobacco in both groups? How many of them were ex-smokers or recent smokers? The mean pack-year given in Table 1 demonstrated how many patients?

Also, in Table 2, transplant age (months) was given as mean 54.1 ± 29.2 for entire, mean 42.5 ± 18.2 for subjects with positive end points, mean 38 ± 13.5 for patients with negative end points. It should be corrected.

We have no conflict of interest to declare.

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REFERENCES